Influence of polycythemia on blood volume and thermoregulation during exercise-heat stress

MICHAEL N. SAWKA, RICHARD C. DENNIS, RICHARD R. GONZALEZ, ANDREW J. YOUNG, STEPHEN R. MUZA, JAMES W. MARTIN, C. BRUCE WENGER, RALPH P. FRANCESCONI, KENT B. PANDOLF, AND C. R. VALERI
US Army Research Institute of Environmental Medicine, Natick 01760-5007; and Naval Blood Research Laboratory/Boston University School of Medicine, Boston, Massachusetts 02118

SAWKA, MICHAEL N., RICHARD C. DENNIS, RICHARD R. GONZALEZ, ANDREW J. YOUNG, STEPHEN R. MUZA, JAMES W. MARTIN, C. BRUCE WENGER, RALPH P. FRANCESCONI, KENT B. PANDOLF, AND C. R. VALERI. *Influence of polycythemia on blood volume and thermoregulation during exercise-heat stress.* J. Appl. Physiol. 62(3): 912–918, 1987.—We studied the effects of autologous erythrocyte infusion on blood volume and thermoregulation during exercise in the heat. By use of a double-blind design, nine unacclimated male subjects were infused with either 600 ml of a NaCl-glucose-phosphate solution containing a ≈50% hematocrit ($n = 6$, reinfusion) or 600 ml of this solution only ($n = 3$, saline). A heat stress test (HST) was attempted ≈2-wk pre- and 48-h postinfusion during the late spring months. After 30 min of rest in a 20°C antechamber, the HST consisted of a 120-min exposure (2 repeats of 15 min rest and 45 min treadmill walking) in a hot (35°C, 45% rh) environment while euhydrated. Erythrocyte volume (RCV, ^{51}Cr) and plasma volume (PV, ^{125}I) were measured 24 h before each HST, and maximal O_2 uptake ($\dot{V}O_{2\,max}$) was measured 24 h after each HST. Generally, no significant effects were found for the saline group. For the reinfusion group, RCV (11%, $P < 0.01$) and $\dot{V}O_{2\,max}$ (11%, $P < 0.05$) increased after infusion, and the following observations were made: *1*) the increased RCV was associated with a reduction in PV to maintain the same blood volume as during the preinfusion measurements; *2*) polycythemia reduced total circulating protein but did not alter F-cell ratio, plasma osmolality, plasma protein content, or plasma lactate at rest or during exercise-heat stress; *3*) polycythemia did not change the volume of fluid entering the intravascular space from rest to exercise-heat stress; and *4*) polycythemia tended to reduce the rate of heat storage during exercise-heat stress.

blood "doping"; blood reinfusion; euhydration; heat storage; plasma volume; erythrocyte volume; temperature regulation

BUICK ET AL. (2) conclusively demonstrated that acute polycythemia improves an individual's submaximal and maximal exercise performance in a comfortable temperature environment. Subsequent investigators have confirmed these findings in comfortable temperature normoxic (10, 13, 14) as well as hypoxic (14) environments. These studies used erythrocyte freeze preservation (24, 25) for autologous reinfusion (at least 2 units) following the reestablishment of normocythemia (8). The physiological mechanism believed primarily responsible for the improved exercise performance is increased arterial O_2 content (8); however, it is possible that blood volume expansion may also contribute to these ergogenic effects (10, 20). Blood volume measurements (from independent measurements of plasma and erythrocyte volume) have not been obtained during previous erythrocyte reinfusion studies, but Kanstrup and Ekblom (10) have measured erythrocyte volume (^{51}Cr). Based on erythrocyte volume measurements, these investigators calculated that blood volume had increased after erythrocyte reinfusion (10). Likewise, two recent animal studies (one measured only plasma volume and the other measured both erythrocyte and plasma volume) also support the concept of an expanded blood volume after erythrocyte infusion (20, 27).

The influence of acute polycythemia on thermoregulatory responses during exercise-heat stress has not been studied. However, there are several reasons why erythrocyte infusion may be beneficial to individuals performing exercise in the heat. During exercise-heat stress, core temperature increases as a consequence of the metabolic and environmental heat load. To minimize these core temperature changes, vasomotor adjustments occur to increase skin blood flow and dilate superficial veins. These adjustments facilitate sensible and insensible heat loss but also displace a portion of the central blood volume to the cutaneous vasculature. Under conditions of combined exercise-heat stress, a competition may exist between the circulatory requirements of metabolically active skeletal muscle and the cutaneous vasculature (15, 17). Eventually this competition can compromise cardiac output as well as heat dissipation (15, 17). Therefore, an intervention which increases arterial O_2 content as well as blood volume might improve exercise-heat performance. Increased arterial O_2 content will improve muscle oxygenation at any level of muscle blood flow, and an expanded blood volume will allow a higher stroke volume and cardiac output. Finally, most investigators believe that during exercise core temperature responses are coupled to relative exercise intensity (4, 16). Erythrocyte reinfusion has been shown to increase maximal aerobic power (2, 10, 13, 14, 23); therefore, the relative exercise intensity and thus core temperature might be lower during exercise at a given O_2 uptake level.

In the present study, we examined the effects of erythrocyte reinfusion on blood volume and thermoregulation

during exercise in the heat. The information gathered should help clarify physiological mechanisms responsible for the ergogenic effects of erythrocyte reinfusion.

METHODS

Subjects. Nine fit male volunteers from the 10th Special Forces Group (Ft. Devens, MA) participated in this investigation. Five additional subjects volunteered and had phlebotomies but were transferred during the subsequent 7 mo or were unavailable for testing. These subjects were all members of the same team and therefore were exposed to similar physical activity, environmental extremes, and diet throughout the study. The subjects were divided into a reinfusion and a saline group. The reinfusion group ($n = 6$) had a mean (±SD) age of 30 ± 7 yr (including the 43-yr-old platoon sergeant), weight of 79 ± 9 kg, surface area-to-mass ratio of 254 ± 14 $cm^2 \cdot kg^{-1}$, and percent body fat of 15 ± 5. The saline group ($n = 3$) had a mean (±SD) age of 22 ± 1 yr, weight of 83 ± 20 kg, surface area-to-mass ratio of 246 ± 26 $cm^2 \cdot kg^{-1}$, and percent body fat of 15 ± 4.

Protocol. During the late fall and early winter months, 2 units of blood were removed by phlebotomy from each subject. A minimum of 6 wk separated the removal of each blood unit. During the subsequent spring months, the experimental portion of the study was completed. Initially, the subjects were familiarized with the test procedures, their percent body fat was determined by hydrostatic weighing, and they completed practice exercise tests. Several days prior to pretesting, the subjects' erythrocyte volume and plasma volume were measured. The pretesting included a maximal aerobic power test and a heat stress test, which were completed on separate days. Approximately 2 wk (range 10–17 days) later each subject received an infusion. The reinfusion group received ~600 ml of a sodium chloride-glucose-phosphate solution containing a ~50% hematocrit (autologous erythrocytes), whereas the saline group received ~600 ml of the sodium chloride-glucose-phosphate solution only. Erythrocyte volume and plasma volume was measured 24-h postinfusion, the heat stress test was attempted 48-h postinfusion, and a maximal aerobic power test was completed 72-h postinfusion.

Blood storage, infusion, and erythrocyte volume and plasma volume measurements were conducted at the Naval Blood Research Laboratory. After each phlebotomy, the blood was separated into its erythrocyte and plasma components, and the erythrocytes were frozen with 40% (wt/vol) glycerol and stored at −80°C (24, 25). For the reinfusion group, ~600 ml of autologous erythrocytes in a sodium chloride-glucose-phosphate solution were administered over a 1-h period. The frozen cell component was thawed and washed to reduce the glycerol concentration to <1%. The erythrocyte O_2 transport function was determined from the erythrocyte 2,3-diphosphoglycerate (2,3-DPG), ATP, and the in vitro hemoglobin O_2 half-saturation pressure (P_{50}) measurements (26). For the saline group, a similar time period was used to administer the sodium-chloride-glucose-phosphate solution. During the infusion sessions, the subjects were blindfolded and wore earphones. Neither the subjects nor the investigators at the US Army Research Institute of Environmental Medicine were aware of the identity and size of the saline and reinfusion groups.

The maximal aerobic power and heat stress tests were conducted at the US Army Research Institute of Environmental Medicine. Each subject's maximal aerobic power was determined by a progressive-intensity continuous-effort treadmill test. The warm-up bout consisted of 4 min of walking (1.56 $m \cdot s^{-1}$) at a 4% treadmill grade. The subjects then ran (3.13 $m \cdot s^{-1}$) continuously at an initial grade of 5% with 2.5% increments every 2 min. Established criteria were employed for determination of maximal O_2 uptake (22). These tests were conducted in a comfortable (20°C ambient temperature, 40% rh) environment.

The heat stress tests (HSTs) were conducted in a hot (35°C ambient temperature, 45% relative humidity) environment. This environment was selected to enable both insensible as well as some sensible heat exchange. Each HST was 120 min (2 repeats of 15 min rest and 45 min exercise) in duration. During exercise, subjects walked (1.56 $m \cdot s^{-1}$) on an inclined (6% grade) treadmill, and during the rest periods they were weighed and rehydrated with spring water to maintain their initial body weight (i.e., euhydration). The subjects wore only shorts and tennis shoes. At least 10 days separated the pre- and postinfusion HSTs to minimize any partial acclimation from the initial heat exposure.

Measurements. Electrocardiogram was obtained with chest electrodes (CM5 placement) and radiotelemetered to an oscilloscope-cardiotachometer unit (Hewlett-Packard). During the maximal aerobic power tests, an automated system (Sensormedics Horizon MMC) was used to measure O_2 uptake. During the HSTs, the respiratory gases were collected in 150-liter Douglas bags. The volume of expired gases was measured with a Tissot gasometer, and the O_2 and CO_2 concentrations were measured with an electrochemical O_2 analyzer (Applied Electrochemistry S-3A) and an infrared CO_2 analyzer (Beckman LB-2), respectively.

During the HSTs, core temperature (T_{co}) measurements were obtained from both the rectum and esophagus. Rectal temperature was measured from a thermistor inserted ~10 cm beyond the anal sphincter, and esophageal temperature was measured from a thermistor placed in a catheter at heart level. Unfortunately, two subjects from the saline group were unable to swallow the esophageal thermistor. Skin temperatures were obtained with a three-point thermocouple skin harness (chest, calf, and forearm), and mean weighted skin temperature ($\bar{T}_{sk}$) was calculated (19). The dew-point temperature of the upper arm was continuously measured by an automatic sensor (9). Body weights were determined on a K-120 Sauter precision electronic balance (accuracy ± 10 g). Total body sweat rates ($\dot{m}_{sw}$) were calculated from nude body weight loss adjusted for water intake and urine output. The rate of total body heat storage (S) was calculated by the equation of $S = \Delta\bar{T}_b \cdot \Delta t^{-1} \cdot 0.97 \cdot m_b \cdot A_D^{-1}$, where $\Delta\bar{T}_b$ (11) is the change in mean body temperature ($\Delta\bar{T}_b = 0.9\ \Delta T_{co} + 0.1\ \Delta\bar{T}_{sk}$), Δt is change in

time, 0.97 is specific heat constant ($W \cdot h \cdot kg^{-1} \cdot °C^{-1}$), m_b is body mass (kg), and A_D is body surface area (m^2). Arm sensible (radiative and convective, $R + C$) heat exchange was determined by using the sum of arm convective heat transfer coefficients (h_{cj}, $W \cdot m^{-2} \cdot °C^{-1}$) and a linear radiation heat transfer coefficient (h_{rj}, taken as 4.4 to 4.7 $W \cdot m^{-2} \cdot °C^{-1}$) multipled by the gradient between arm skin temperature and the ambient temperature. We used an average local area convective heat transfer of 7.3 $W \cdot m^{-2} \cdot °C^{-1}$ as determined by naphthalene sublimation (12).

Venous blood samples were collected from an indwelling Teflon catheter placed within a superficial forearm vein. Patency was maintained with heparinized saline; the catheter (2 ml of dead space) was flushed with 4 ml of blood before each 5-ml sample was obtained. Blood samples taken at rest were obtained while all the subjects stood (for 20 min prior to sampling) in the antechamber (20°C ambient temperature, 40% relative humidity), and exercise blood samples were obtained 30-min into each exercise bout while the subjects continued to walk. Triplicate measurements were made for all blood variables. Automated systems were used for hemoglobin (Hemoglobinometer, Coulter Electronics) and plasma lactate (model 23 lactate analyzer, Yellow Springs Instrument). Plasma osmolality was measured with freezing point depression (Osmette A, Precision Systems) and plasma protein concentration was quantitated with refractometer (American Optical). The percent change in plasma volume from rest to exercise was calculated from the appropriate hemoglobin and hematocrit values (5). Erythrocyte volume (RCV) and plasma volume (PV) at rest were measured by the radioactively labeled chromium- (^{51}Cr) and iodine-labeled (^{125}I) albumin methods (26), respectively. The plasma volumes during exercise were calculated by adjusting the measured plasma volume at rest by the appropriate relative percent change in plasma volume. The F-cell ratio (3) was calculated from the ratio of overall hematocrit (H_o) to the peripheral venous hematocrit (not corrected for trapped plasma). The overall hematocrit was calculated as

$$H_o = \frac{RCV}{RCV + PV}$$

Statistical analyses. Means ± SD, simple regression, and repeated measures analyses of variance followed by Bonferroni (7) procedures were used. Statistical significance was tested at the $P < 0.05$ level. It was not our intent to make direct comparisons between the saline and reinfusion groups; therefore, an unbalanced experimental design was selected. The saline group was used to control for the influence of partial heat acclimation during the study as well as to provide an index of pre- to postinfusion measurement variability.

RESULTS

Infusion. Table 1 provides the subjects' resting hematological measurements for pre- and postinfusion. For the reinfusion group, there was increased ($P < 0.05$) erythrocyte volume by 11%, hemoglobin by 10%, and hematocrit by 12% from pre- to postreinfusion. Figure 1 indicates that the increased erythrocyte volume (222 ml) was associated ($r = -0.72$) with a reduced plasma volume (265 ml) from pre- to postreinfusion. Neither blood volume nor F-cell ratio was altered by reinfusion. For the reinfusion group, no differences were found for red cell 2,3-DPG, 15.3 ± 2.3 to 14.4 ± 2.0 $\mu mol \cdot g\ Hb^{-1}$; ATP, 4.2 ± 0.5 to 4.2 ± 0.8 $\mu mol \cdot g\ Hb^{-1}$; and P_{50}, 27 ± 2 to 27 ± 1 Torr from pre- to postreinfusion. Since the actual transfused erythrocyte volume was 299 ± 17 ml, the percent survival rate was 74 ± 19%. However, calculation of the survival rate from the transfused erythrocyte volume is known to result in large experimental error. For the saline group, there was decreased ($P < 0.05$) erythrocyte volume by 3% and blood volume by 4% from pre- to postinfusion.

Maximal exercise. For the reinfusion group, maximal O_2 uptake increased ($P < 0.05$) by 11% from pre- (4.280 $l \cdot min^{-1}$) to post- (4.753 $l \cdot min^{-1}$) reinfusion. Neither heart rate nor ventilatory equivalent of O_2 were altered by reinfusion. For the saline group, heart rate, ventilatory equivalent of O_2, and maximal O_2 uptake were not altered by infusion.

Heat stress tests. All nine subjects completed (120 min) each HST. Table 2 provides the subjects' physiological responses to the HSTs. For the reinfusion group, metabolic rate and mean skin temperature were not altered, but final exercise heart rate was reduced ($P < 0.05$) from pre- to postreinfusion. No differences were found for final exercise rectal temperature (38.7 ± 0.6 to 38.5 ± 0.2°C) nor final exercise esophageal temperature (38.3 ± 0.5 to 38.0 ± 0.1°C) from pre- to postreinfusion. Heat storage as calculated from rectal temperature changes was lower ($P < 0.05$) for the first but not the second exercise bout during the postreinfusion HSTs. The rate of heat storage as calculated from esophageal temperature changes tended to be lower ($P > 0.05$) postreinfusion and is depicted in Fig. 2. It can be noted that 11 of 12 observations (6 subjects × 2 exercise bouts) demonstrated lower values postreinfusion. Table 3 provides the subjects' steady-state heat exchange during the HSTs. For the reinfusion group, total body sweat rate, evaporative heat loss through the skin (arm E_{sk}), and arm ($R + C$) were not altered from pre- to postreinfusion.

For the reinfusion group, plasma volume was decreased from the pre- to postreinfusion HSTs. Figure 3 presents the reinfusion group's plasma volume and percentage change in plasma volume from rest to exercise during the HSTs. The percent change in plasma volume from rest to exercise was greater ($P < 0.01$) postreinfusion, but the absolute fluid volume that moved from the interstitial to the intravascular space was nearly identical (~190 ml) pre- and postreinfusion. Table 4 presents the plasma osmolality, plasma lactate, plasma protein content, and total circulating protein during the HSTs. For the reinfusion group, plasma osmolality, plasma lactate, and plasma protein content were not altered from pre- to postreinfusion. However, total circulating protein was lower ($P < 0.01$) postreinfusion.

For the saline group, none of the variables listed in Tables 2–4 nor plasma volume and percent change in

TABLE 1. *Influence of erythrocyte or saline infusion on hematological variables at rest*

Reinfusion	Erythrocyte Volume, liters	Plasma Volume, liters	Blood Volume, liters	Hemoglobin, g·dl^{-1}	Venous Hematocrit, %	F-Cell Ratio
Pre	2.079±0.258	3.674±0.285	5.753±0.425	13.9±1.1	42±3	0.89±0.06
Post	2.301±0.234	3.409±0.238	5.710±0.451	15.3±1.1	47±2	0.87±0.03

Values are means ± SD; $n = 6$ subjects. F-cell ratio = H_o/H_v, where H_o is overall hematocrit and H_v is venous hematocrit.

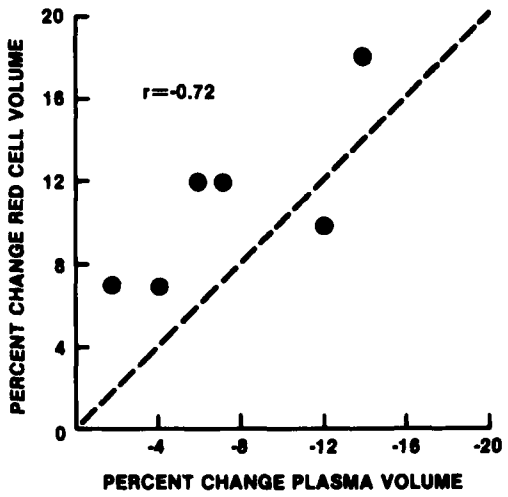

FIG. 1. Relationship between percent change in erythrocyte volume and percent change in plasma volume after erythrocyte reinfusion.

plasma volume from rest to exercise were altered by infusion.

DISCUSSION

It is generally accepted that several recent Summer Olympic medal winners have used blood infusions (homologous and autologous) or "blood doping" as an ergogenic aid. These athletes participated in endurance events that required high maximal aerobic power and made considerable thermoregulatory demands for heat dissipation. Although we do not advocate the use of blood doping as an ergogenic aid for athletic competition, autologous erythrocyte infusion provides a powerful tool to further our understanding of physiological control mechanisms in response to exercise-heat stress. Although blood doping for athletic competition is unsanctioned and considered unethical, it can increase maximal aerobic power provided that specific blood handling and infusion procedures are used (8). By employing these procedures, our subjects had an ~11% increment for their O_2-carrying capacity and subsequent maximal aerobic power. The magnitude of these increases are consistent with those reported by others employing similar procedures (2, 10, 13, 14, 23).

Our data indicate that acute polycythemia results in a compensatory reduction in plasma volume (Fig. 1). The reinfused subjects had an increased erythrocyte volume and decreased plasma volume of 222 and 265 ml, respectively. Interestingly, the saline group manifested a decreased erythrocyte volume and plasma volume from pre- to postinfusion. This observation raises the possibility that systematically decreased postreinfusion measurements may have masked a slight increase in blood volume for the reinfusion group. Bentley and Lewis (1) independently measured erythrocyte volume (^{51}Cr) and plasma volume (^{125}I) in 130 patients with polycythemia and a variety of other hematological disorders. They found a positive linear relationship ($P < 0.001$) between venous hematocrit and total blood volume in patients with venous hematocrits of >50%, but in patients with lower hematocrits no relationship was found between these variables. Bentley and Lewis (1) also observed that for individuals with venous hematocrits of ~40% or less there is an inverse ($r = -0.75$; $P < 0.001$) relationship between venous hematocrit and plasma volume. Our subjects had initial venous hematocrits of 42% (range 37–45%); therefore, the compensatory reduction in plasma volume for the increased erythrocyte volume ($r = -0.72$) was consistent with the clinical data from Bentley and Lewis (1). It seems possible that the plasma volume responses to erythrocyte infusion may somehow be dependent on the initial preinfusion hematocrit.

Valeri and Altschule (26) have reported that an erythrocyte transfusion can increase plasma volume in trauma patients. As erythrocytes do not exert an in vitro oncotic pressure, they reported that the expanded plasma volume from erythrocyte transfusion was mediated by a mobilization of interstitial albumin into the intravascular space (25–27). Of note is that their protein-mediated plasma volume expansion in trauma patients is nearly identical to that mechanism contributing to heat-acclimation hypervolemia (21). Interestingly, the patients were wounded servicemen who had been transported from Southeast Asia, usually within the preceding 2 wk. These individuals were probably heat acclimated from living in a warm climate. Our subjects were unacclimated to heat; in fact, they had participated in cold-weather training before and during the study. Perhaps, if heat-acclimated subjects were reinfused, their greater availability of extravascular protein (21) might have allowed a plasma volume expansion.

During the postreinfusion HSTs, the reinfused subjects had a reduced (~7%) plasma volume with the same blood volume as in the prereinfusion HSTs. This reduced plasma volume did not affect the absolute magnitude (~190 ml) of hemodilution resulting from the transition from rest to exercise. Therefore, plasma volume per se does not exert an effect independent of blood volume on vascular fluid shifts during exercise-heat stress. This observation is of interest, but not surprising, since the transcapillary osmotic, oncotic, and probably hydrostatic pressures were not different from pre- to postreinfusion. On the other hand, several investigators (6, 10) have shown that manipulation of plasma volume to change

TABLE 2. *Influence of erythrocyte or saline infusion on physiological measurements during heat stress-exercise tests*

Reinfusion	Metabolic Rate, $W \cdot m^{-2}$	Heart Rate, $beats \cdot min^{-1}$		T_{sk}, °C		S, T_{re}, $W \cdot m^{-2}$		S, T_{es}, $W \cdot m^{-2}$	
		Ex-1	Ex-2	Ex-1	Ex-2	Ex-1	Ex-2	Ex-1	Ex-2
Pre	358±25	139±15	145±19	33.5±0.4	33.3±0.5	72±11	29±11	65±11	72±39
Post	349±28	132±15	141±13	33.9±0.9	34.0±0.8	63±9	29±5	55±15	43±5

Values are means ± SD; $n = 6$ subjects. Heat stress conditions: 35°C, 45% rh. $\bar{T}_{sk}$, mean skin temperature; S, rate of heat storage; T_{re}, rectal temperature; T_{es}, esophageal temperature; Ex-1 and Ex-2, exercise bouts 1 and 2, respectively.

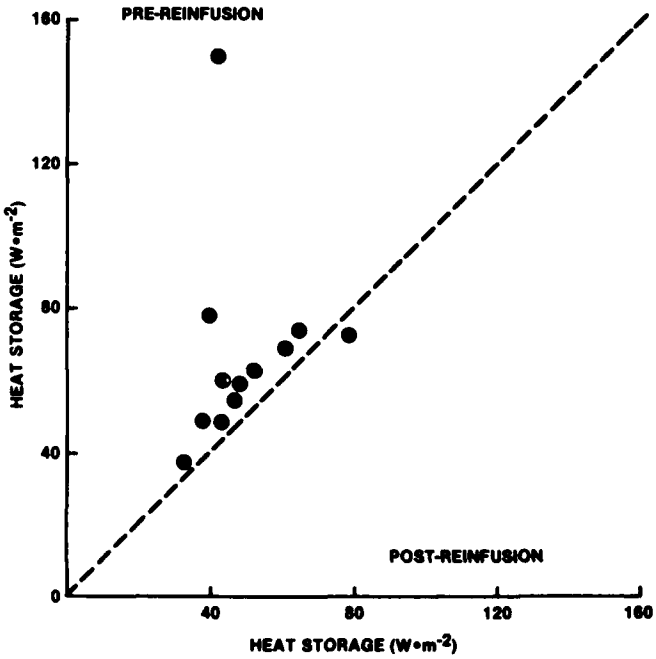

FIG. 2. Individual data plots of pre- to postreinfusion values for heat storage (calculated from esophageal temperature) during heat stress tests.

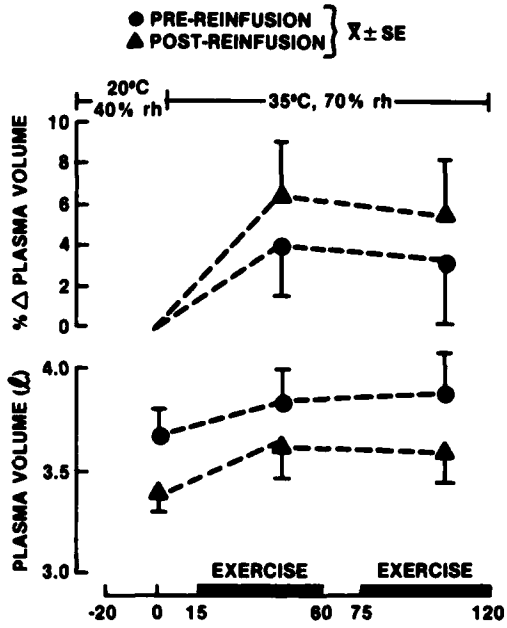

FIG. 3. Plasma volume and percent change in plasma volume from rest during the pre- and postreinfusion heat stress tests.

TABLE 3. *Influence of erythrocyte or saline infusion on heat exchange measurements during heat stress-exercise tests*

Reinfusion	Arm $(R + C)$, $W \cdot m^{-2}$		Arm E_{sk}, $W \cdot m^{-2}$		Total Body Sweat Rate, $g \cdot m^{-2} \cdot h^{-1}$	
	Ex-1	Ex-2	Ex-1	Ex-2	Ex-1	Ex-2
Pre	−12.0±12.0	−0.6±8.0	240±34	375±29	386±52	581±46
Post	−14.9±6.2	−10.1±5.9	240±38	359±32	384±58	559±49

Values are means ± SD; $n = 6$ subjects. Heat stress conditions: 35°C, 45% rh. Ex-1 and Ex-2, exercise bouts 1 and 2, respectively. Arm sensible (radiative and convective, $R + C$) and insensible (evaporative from skin, E_{sk}) heat exchange values represent steady-state (final exercise) values. Negative values represent a heat gain by the body.

blood volume will alter vascular fluid shifts during exercise. Finally, the ~7% reduction in plasma volume is similar in magnitude to the reduction associated with the hypovolemia during moderate hypohydration (19). As a result, it might be interesting to examine the hematological responses of reinfused subjects who had the additional challenge of hypohydration during exercise-heat stress.

We are the first to examine the influence of acute polycythemia on thermoregulation during exercise-heat stress. Our data indicate that polycythemia provides a small thermoregulatory advantage for euhydrated nonheated-acclimated individuals. The rate of heat storage values, as calculated from esophageal temperatures, were lower postreinfusion for 11 of 12 observations. Heat storage values as calculated from rectal temperature were only lower during the first exercise bout for the reinfusion group. For the saline group, a tendency for a greater rate of heat storage was evident during the postinfusion HST. The relative contributions of insensible and sensible heat exchange for the reduced rate of heat storage after erythrocyte reinfusion remain unclear. Total body sweat rate, steady-state arm E_{sk}, and steady-state arm $(R + C)$ values did not differ between the two conditions. These measurements may not have been sufficiently sensitive to detect such small differences; alternatively, both insensible and sensible heat loss from the arm may not always follow changes from other body regions (6, 18). Finally, the improved effector responses for heat loss may have occurred at the onset of exercise (lower threshold responses) and therefore would not be evident in the steady-state measurements. During steady-state conditions, by definition, heat loss should be roughly equal to heat gain.

We can hypothesize a potential physiological mechanism for improved sensible heat exchange after erythro-

TABLE 4. *Influence of erythrocytes or saline infusion on plasma osmolality, lactate, and protein and total circulating protein during heat stress-exericse tests*

Reinfusion	Osmolality, mosmol·kg^{-1}			Lactate, mmol·l^{-1}			Protein Content, g·dl^{-1}			Total Circulating Protein, g		
	Rest	Ex-1	Ex-2	Rest	Ex-1	Ex-2	Rest	Ex-1	Ex-2	Rest	Ex-1	Ex-2
Pre	285±2	288±2	289±3	1.1±0.3	1.3±0.3	1.1±0.5	8.1±0.7	7.8±0.5	8.0±0.5	299±34	298±37	308±29
Post	287±3	288±3	289±4	1.2±0.3	1.5±0.6	1.4±0.4	8.3±0.6	7.8±0.5	8.0±0.5	284±36	283±36	287±36

Values are means ± SD; n = 6 subjects. Heat stress conditions: 35°C, 45% rh. Ex-1 and Ex-2, exercise bouts 1 and 2, respectively.

cyte infusion, namely, that the elevated arterial O_2 content allowed a reduced skeletal muscle blood flow and thus increased cutaneous blood flow at a given cardiac output during submaximal exercise. Welch et al. (29) found that a 10% increase in arterial O_2 content during hyperoxia in humans resulted in a similar decrease in muscle blood flow during submaximal exercise. Likewise, several studies using dogs have reported similar findings of reduced skeletal muscle blood flow when arterial O_2 content was elevated by hyperoxia during submaximal exercise (28, 30).

Our data indicate several new findings concerning acute polycythemia: *1*) the increased erythrocyte volume was associated with a reduction in plasma volume to maintain the same blood volume as during the preinfusion measurements; *2*) polycythemia reduced total circulating protein but did not alter F-cell ratio, plasma osmolality, plasma protein content, or plasma lactate at rest or during exercise-heat stress; *3*) polycythemia did not change the volume of fluid entering the intravascular space from rest to exercise-heat stress; and *4*) polycythemia tended to reduce the rate of heat storage during exercise-heat stress. These results should not be generalized beyond euhydrated subjects who are unacclimated to heat.

Recognition is due to the volunteer members of the 10th Special Forces Group stationed at Ft. Devens, MA, and their leaders for allowing them to participate. Recognition is also due to Ron Carciero, George Cassidy, Holly Feingold, Alan Gray, and Gina Ragno for the collection and processing of the blood for storage. The authors gratefully acknowledge Patricia DeMusis and Dorothy Leader for technical assistance in preparing the manuscript and Tammy Doherty for statistical analyses.

This study was supported in part by the Office of Naval Research Contract N00014-79-0168.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of Army position, policy, or decision, unless so designated by other official documentation.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

Address for reprint requests: M. N. Sawka, US Army Research Institute of Environmental Medicine, Kansas St., Natick, MA 01760-5007.

Received 10 February 1986; accepted in final form 22 September 1986.

REFERENCES

1. BENTLEY, S. A., AND S. M. LEWIS. The relationship between total red cell volume, plasma volume and venous hematocrit. *Br. J. Haematol.* 33: 301–307, 1976.
2. BUICK, F. J., N. GLEDHILL, A. B. FROESE, L. SPIRET, AND E. C. MEYERS. Effect of induced erythrocythemia on aerobic work capacity. *J. Appl. Physiol.* 48: 636–642, 1980.
3. CHAPLIN, H., P. L. MOLLISON, AND H. VETTER. The body/venous hematocrit ratio: its consistency over a wide hematocrit range. *J. Clin. Invest.* 32: 1309–1316, 1953.
4. DAVIES, C. T. M., J. R. BROTHERHOOD, AND E. ZEIDIFARD. Temperature regulation during severe exercise with some observations on effects of skin wetting. *J. Appl. Physiol.* 41: 772–776, 1976.
5. DILL, D. B., AND D. L. COSTILL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37: 247–248, 1974.
6. FORTNEY, S. M., E. R. NADEL, C. B. WENGER, AND J. R. BOVE. Effect of blood volume on sweating rate and body fluids in exercising humans. *J. Appl. Physiol.* 51: 1594–1600, 1981.
7. GLANTZ, S. A. *Primer of Biostatistics.* New York: McGraw-Hill, 1981, p. 87–90.
8. GLEDHILL, N. The influence of altered blood volume and oxygen transport capacity on aerobic performance. *Exercise Sport Sci. Rev.* 13: 75–93, 1985.
9. GRAICHEN, H., R. RASCATI, AND R. R. GONZALEZ. Automatic dew-point temperature sensor. *J. Appl. Physiol.* 52: 1658–1660, 1982.
10. KANSTRUP, I., AND B. EKBLOM. Blood volume and hemoglobin concentration as determinants of maximal aerobic power. *Med. Sci. Sports Exercise* 16: 256–262, 1984.
11. MINARD, D. Body heat content. In: *Physiological and Behavioral Temperature Regulation*, edited by J. D. Hardy, A. P. Gagge, and J. A. J. Stolwijk. Springfield, IL: Thomas, 1970, p. 345–357.
12. NISHI, Y., AND A. P. GAGGE. Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. *J. Appl. Physiol.* 29: 830–838, 1970.
13. ROBERTSON, R. J., R. GILCHER, K. F. METZ, C. J. CASPERSEN, T. G. ALLISON, R. A. ABBOTT, G. S. SKRINER, J. R. KRAUSE, AND P. A. NIXON. Hemoglobin concentration and aerobic work capacity in women following induced erythrocythemia. *J. Appl. Physiol.* 57: 568–575, 1984.
14. ROBERTSON, R. J., R. GILCHER, K. F. METZ, G. S. SKRINER, T. G. ALLISON, H. T. BAHNSON, R. A. ABBOTT, R. BECKER, AND J. E. FALKEL. Effect of induced erythrocythemia on hypoxia tolerance during physical exercise. *J. Appl. Physiol.* 53: 490–495, 1982.
15. ROWELL, L. B. Human cardiovascular adjustments to exercise and thermal stress. *Physiol. Rev.* 54: 75–159, 1974.
16. SALTIN, B., AND L. HERMANSEN. Esophageal, rectal, and muscle temperature during exercise. *J. Appl. Physiol.* 21: 1757–1762, 1966.
17. SAWKA, M. N., R. P. FRANCESCONI, A. J. YOUNG, AND K. B. PANDOLF. Influence of hydration level and body fluids on exercise performance in the heat. *J. Am. Med. Assoc.* 252: 1165–1169, 1984.
18. SAWKA, M. N., R. R. GONZALEZ, L. L. DROLET, AND K. B. PANDOLF. Heat exchange during upper- and lower-body exercise. *J. Appl. Physiol.* 57: 1050–1054, 1984.
19. SAWKA, M. N., A. J. YOUNG, R. P. FRANCESCONI, S. R. MUZA, AND K. B. PANDOLF. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J. Appl. Physiol.* 59: 1394–1401, 1985.
20. SCHUMACKER, P. T., B. GUTH, A. J. SUGGETT, P. D. WAGNER, AND J. B. WEST. Effects of transfusion-induced polycythemia on O_2 transport during exercise in the dog. *J. Appl. Physiol.* 58: 749–758, 1985.
21. SENAY, L. C., JR. Changes in plasma volume and protein content during exposures of working men to various temperatures before and after acclimation to heat: separation of the roles of cutaneous and skeletal muscle circulation. *J. Physiol. Lond.* 224: 61–81, 1972.
22. TAYLOR, H. L., E. R. BUSKIRK, AND A. HENSCHEL. Maximal oxygen intake as an objective measure of cardio-respiratory per-

formance. *J. Appl. Physiol.* 8: 73–80, 1955.
23. THOMSON, J. M., J. A. STONE, A. D. GINSBURG, AND P. HAMILTON. O_2 transport during exercise following blood reinfusion. *J. Appl. Physiol.* 53: 1213–1219, 1982.
24. VALERI, C. R. *Blood Banking and the Use of Frozen Blood Products.* Cleveland, OH: CRC, 1976.
25. VALERI, C. R. New developments in red blood cell preservation using liquid and freezing procedures. *J Clin. Lab. Auto.* 2: 425–439, 1982.
26. VALERI, C. R., AND M. D. ALTSCHULE. *Hypovolemic Anemia of Trauma: the Missing Blood Syndrome.* Boca Raton, FL: CRC, 1981.
27. VALERI, C. R., K. DONAHUE, H. M. FEINGOLD, G. P. CASSIDY, AND M. D. ALTSCHULE. Increase in plasma volume after the transfusion of washed erythrocytes. *Surg. Gynecol. Obstet.* 162: 30–36, 1986.
28. WELCH, H. G., J. P. MULLIN, G. D. WILSON, AND J. LEWIS. Effects of breathing O_2-enriched gas mixtures on metabolic rate during exercise. *Med. Sci. Sports* 6: 26–32, 1974.
29. WELCH, H. G., F. B. PETERSEN, T. GRAHAM, K. KLAUSEN, AND N. SECHER. Effects of hyperoxia on leg blood flow and metabolism during exercise. *J. Appl. Physiol.* 42: 385–390, 1977.
30. WILSON, B. A., AND W. N. STAINSBY. Effects of O_2 breathing on RQ, blood flow, and developed tension in in situ dog muscle. *Med. Sci. Sports* 10: 167–170, 1978.